

100



# UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE  
 United States Patent and Trademark Office  
 Address: COMMISSIONER FOR PATENTS  
 P.O. Box 1450  
 Alexandria, Virginia 22313-1450  
 www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/423,905	04/24/2000	TOHRU TANI	FJN-077	7282

7590 07/15/2004

TESTA HURWITZ & THIBEAULT  
 HIGH STREET TOWER  
 125 HIGH STREET  
 BOSTON, MA 02110

EXAMINER

DUFFY, PATRICIA ANN

ART UNIT	PAPER NUMBER
----------	--------------

1645

DATE MAILED: 07/15/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

**Office Action Summary**

Application No.

09/423,905

Applicant(s)

TANI ET AL.

Examiner

Patricia A. Duffy

Art Unit

1645

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
  - If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
  - If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
  - Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☒ Responsive to communication(s) filed on 9-30-03
- 2a) ☐ This action is **FINAL**.                      2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 2-5, 8-10, 13-16, 19-21 and 24-35 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 2-5, 8-10, 13-16, 19-21 and 24-35 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
- Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All    b) ☐ Some \*    c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

- |   |   |
|---|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)             | 4) <input type="checkbox"/> Interview Summary (PTO-413)                     |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)    | Paper No(s)/Mail Date. _____  |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) | 5) <input type="checkbox"/> Notice of Informal Patent Application (PTO-152) |
| Paper No(s)/Mail Date _____   | 6) <input type="checkbox"/> Other: _____                                    |

## RESPONSE TO AMENDMENT

The amendment filed 9-30-03 has been entered into the record. Claims 2-5, 8-10, 13-16, 19-21 and 24-35 are pending and under examination.

The text of Title 35 of the U.S. Code not reiterated herein can be found in the previous office action.

### *New Rejections Based on Amendment*

Claims 2-5, 8-10, 13-16, 19-21 and 24-35 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a new matter rejection.

As to claims 2-5, 8-10 and 34-35, the claims are drawn to a method for reducing sepsis-associated lethality in a mammal that develops sepsis, the method comprising the step of administering an amount of isolated or purified tissue cytotoxic factor-II (TCF-II) effective to reduce sepsis-associated lethality, wherein TCF-II is administered before the onset of sepsis in the mammal. This concept, as applied to mammals in general does not have written description support in the specification as filed. The specification teaches a rat-cecum puncture model, wherein the individual rats are punctured in the cecum and each rat predictably develops sepsis. There is no written description of deliberately inducing sepsis in the genus of mammals wherein all the mammals predictably develop sepsis, such that the TCF-II can be administered prior to the onset of sepsis as in the rat model of sepsis. There are no circumstances or diseases described in this specification where sepsis is predictably induced in a mammal, outside of the particular rat model disclosed, such that the timing of the administration of the TCF-II can happen

before the onset of sepsis. There is no conception of applying deliberate induction of sepsis in the studied rat model to mammals in general. As such, the broadening of the concept presented for the specific rat model to apply to all mammals is not found to have conception by way of written description in the specification and is therefore deemed new matter. Additionally, claim 34 recites that TCF-II can be administered "at the time of sepsis". This time of administration is not supported by the specification or Examples presented therein as filed.

As to claims 32 and 33, the specification fails to support treatment prior to surgery or at the time of surgery as is now claimed. The treatment modalities as set forth in the examples provide for the combined treatment regimen of administering prior and after cecum puncture. The specification fails to support the now specifically claimed subgenera s of prior, after or at the time of surgery.

As to claims 13-16, 19-21, and 28-30 the concept of a "trigger" is set forth at page 2 of the specification that teaches "Transfer of bacteria or products secreted by them from the intestine is called bacterial translocation and is noticed as a trigger of inducing sepsis." This passage defines transfer of bacteria or products secreted by them as the trigger for sepsis. This passage does not define the concept of "triggers" as used in claims 13 because "triggers" are not defined as the secondary events that may or may not cause translocation. The trigger of sepsis is taught by the specification as the translocation of bacteria or bacterial products. As such, this passage does not support the claims for A method for reducing lipopolysaccharide (LPS)-induced bacterial translocation in the intestine in a mammal exposed to a trigger for LPS-induced bacterial translocation, as now claimed. Further, clearly according to the claims LPS-induces bacterial translocation. Is therefore LPS not the trigger inducing event ? Applicants are mixing concepts from the model of Example 2, with this passage to form a third concept with language that does not have conception by way of written description in the specification (triggers for LPS-induced bacterial translocation). In Example 2, LPS is

intravenously administered and bacterial translocation is measured in treated and untreated rats. As such, intravenous LPS in this case would be seen the immediate cause of bacterial translocation, the bacterial translocation the trigger of sepsis. There is no additional trigger described or disclosed in the specification. In example 2, LPS is the presupposing induction event for bacterial translocation. There are no disclosed triggers for LPS-induced bacterial translocation. There is no concept of triggers for LPS-induced bacterial translocation. These methods lack conception by way of written description in the specification as filed. There is no conception of LPS-induced bacterial translocation in mammals in general. There is no teaching that broadens this teaching to the genus of mammals using "triggers", which are not described in the specification.

As to claims 24-27, the claim recites a method for reducing sepsis-associated lethality in a mammal exposed to "a trigger for sepsis". The specification teaches at page 2, the concept of a "trigger". "Transfer of bacteria or products secreted by them from the intestine is called bacterial translocation and is noticed as a trigger of inducing sepsis." This passage defines transfer of bacteria or products secreted from the intestine as the "trigger for sepsis". The specification does not teach that mammals are "exposed" to a trigger for sepsis, rather that the process of translocation is the trigger for sepsis. How does one expose one to a transfer of bacteria or products from the intestine? This concept is not conveyed by the specification as originally filed and is certainly not conveyed by the concept of a trigger as described on page 2 of the instant specification. There is no descriptions of a trigger for sepsis outside of that of page 2. As such, the specification fails to support the now claimed concept of invention in regard to "triggers" for sepsis. The trigger for sepsis is clearly noticed in the specification as the bacterium or product thereof. The language such as "trigger for LPS-induced bacterial translocation" can not be said to be either explicitly or implicitly described in this specification. Additionally, claim 27 recites that TCF-II can be administered "at the

time of exposure of the mammal to the trigger for sepsis". This time of administration is not supported by the specification or Examples presented therein as filed.

Claims 2-5, 8-10 and 34-35 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method for reducing sepsis-associated lethality in a cecum-punctured rat that develops sepsis, comprising administering an amount of isolated or purified tissue cytotoxic factor-II (TCF-II) effective to reduce sepsis associated lethality, wherein the TCF-II is administered prior to the cecum being punctured and the onset of sepsis, it does not reasonably provide enablement for methods for reducing the lethality in a mammal that develops sepsis by administering an amount of isolated or purified tissue cytotoxic factor-II (TCF-II) effective to reduce sepsis associated lethality, wherein the TCF-II is administered prior to the onset of sepsis. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

The claims are drawn to a method for reducing sepsis-associated lethality in *a mammal that develops sepsis*, the method comprising the step of administering an amount of isolated or purified TCF-II effective to reduce sepsis-associated lethality, wherein TCF-II is administered before the onset of sepsis in *the mammal*. This claim as currently constructed requires prior knowledge and absolute prediction of what mammals will get sepsis and also requires administration at a time before the onset of sepsis to *the mammal* (i.e. the mammal that develops sepsis). Outside of deliberate induction of sepsis in the rat cecum puncture model of Examples 1 of the specification, the specification does not teach how to identify members of the genus of mammals, including humans, that absolutely develop sepsis, before the onset of sepsis thereof in that mammal. In other words, to be enabling the specification must be able to predictably and reproducibly identify those mammals that will develop sepsis (i.e. in the future). The art and the specification define

sepsis as the presence of various pathogenic organisms or their toxins in the blood or tissues. Sepsis is clearly induced by a pathogenic bacterial infection and not induced by chemotherapy, radiotherapy, dialysis etc. as asserted in the specification at pages 1-2 of the specification. While the specification describes "inducers of sepsis" such as radiotherapy etc, it is clear from the art, that the known sole inducer of sepsis is an active bacterial infection. As such, any of the treatments described at pages 1-2 are not innately causative of sepsis. Chemotherapy does not necessarily lead to sepsis. Cancer does not necessarily lead to sepsis. Cerebrovascular disease does not necessarily lead to sepsis. While sepsis may be a consequence of the suppression of the immune system in some individuals, all individuals that have cancer, cerebrovascular disease, undergo chemotherapy or have surgery, do not necessarily develop sepsis. Sepsis "may" or "may not" develop these individuals. These procedures, diseases only put an individual at risk of developing sepsis. Even for the most invasive of these risks, surgery, the art teaches that only a percentage of individuals undergoing surgery develop sepsis and that there is no established predictor of those patients that develop sepsis and those that do not. As such, neither the art, nor this specification teach how to predictably and reproducibly identify those mammalian patients who will develop sepsis in order to treat those same individuals before they develop sepsis. At best, the art teaches that individuals that are "at risk" can be identified (Holzheimer et al, Infection Control and Hospital Epidemiology 18(6):449-456, 1997). Risk is a probability, and even Holzheimer et al, does not provide for any category of human that absolutely predicts those individuals who will develop sepsis. Further, Cainzos et al (Hepato-Gastroenterology, 44(16):959-967, 1997) teaches that even surgery does not 100% predict patients developing sepsis and teach that using conventional surgical procedures in humans, only 7.5% developed postoperative septic complications. As such, surgery *per se* in humans (i.e. as representative of the genus mammals) is not absolutely predictive of sepsis. The claims are seen to require absolute predictability because in order to perform the method of the claimed invention you must

be able to identify that population of mammals that "develops sepsis" and treat the mammal prior to the onset of sepsis. The only population of for which one can predict development of sepsis is the rat cecum puncture model described in Examples 1 and 2 of the specification. No other animal population or human population is described or identified in the specification that develops sepsis and the art teach that at best only "risk" or probability can be assessed. There exists no predictable method of identifying only those humans, veterinary mammals etc which will develop sepsis, due surgery, to any of the immunosuppressive treatments, cancer, AIDS, collagen disease, renal insufficiency, hepatic disease, cerebrovascular disease, diabetes, aged persons, or immature infants that will develop sepsis, such that intervention (i.e. the administration of TCF-II) can occur prior to the onset of sepsis in the mammal. Neither the art nor this specification teach methods for identification of this mammal population. Further, animal models of inhibition of sepsis do not predict success in humans. Karazi et al (International Journal of Clinical Practice, 51(4) :232-237, 1997) teach that the pathogenesis of sepsis involves not only microbial toxins but also activated host inflammatory mediators. Karazi et al teach that although, the modulation of inflammatory response has been demonstrated in animal models of sepsis, it has failed to improve survival or otherwise show efficacy in humans. As such, it is clear that the animal model presented is not predictive of success in humans, which is included in the scope of mammals and humans are the primary target of administration in the specification (see pages 1-2 of the specification). In view of the lack of teaching in the specification and in the art of how to predict those individuals who will get sepsis, and humans in particular, which is required by the claim in order to administer the TCF-II prior to the onset of sepsis in the mammal. The claim is not drawn to a prophylaxis of sepsis by treating before the onset of sepsis. All the treated individuals of this claim will go on to develop sepsis, according to the preamble of the claim. In view of the lack of teaching identifiable mammalian populations, other than the rat cecum puncture model, the lack of absolute correlation of even invasive surgery with



sepsis and the lack of predictability of an animal model with therapeutic effect in humans, it would require undue experimentation to practice the scope of the invention as claimed and the claims should be so limited.

Claims 16, 24, 28, 30 and 35 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

As to claim 16, the claim recites purifying an amount effective to prevent sepsis, however, this lack correlation with the body of the claim drawn to reducing LPS-induced bacterial translocation. Applicants should clarify this step for the record.

As to claim 24, the claim recites that TCF-II is administered within 48 hours of exposure to the trigger. This claim clearly encompasses administration before or after the trigger and as such renders the scope of the invention unclear. The examiner suggest that the language be amended to recite that the TCF-II is administered within 30 hours before exposure to the trigger would obviate the confusion.

As to claim 28, the claim recites "...after exposure of the mammal to the trigger for sepsis.", which lacks antecedent basis in independent claim 28.

As to claim 30, the claim recites that TCF-II is administered within 30 hours of exposure to the trigger. This claim clearly encompasses administration before or after the trigger and as such renders the scope of the invention unclear. The examiner suggest that the language be amended to recite that the TCF-II is administered within 30 hours before exposure to the trigger would obviate the confusion.

As to claim 35, the claim recites that TCF-II is administered within 48 hours of sepsis. This claim clearly encompasses administration before or after the trigger and as such renders the scope of the invention unclear. The examiner suggest that the language be amended to recite that the TCF-II is administered within 48 hours before sepsis, would obviate the confusion.

Art Unit: 1645

As to claims 2-5, 8-10, 13-16, 19-21, 24-30 and 32-35, the specification provides written description support for times of administration of TCF-II. The dosing regimens set forth therein specifically recite administration prior to and after. These examples do not provide conception for the administration of only before (prior to) or only after or at the time. The specification does not convey the concept of the now claimed subgenus of times of administration. It cannot be said that a subgenus is necessarily described by a genus encompassing it and a species upon which it reads. In re Smith 173 USPQ 679, 683 (CCPA 1972). See MPEP 2163.05(b). In this case, the genus is administration, the species is the administration both before and after. There is no conception by way of written description that Applicants had conceived at the time of invention the administration prior to in the absence of after or conversely administration in the absence of prior to the trigger for sepsis, LPS-induced bacterial translocation, surgery etc.. Thus, the Examples which provide for specific administration regimens are not seen to support the now claimed subgenus of regimens.

Claims 13-16, 19-21, and 28-30 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The claims are drawn to a method for reducing lipopolysaccharide (LPS)-induced bacterial translocation in the intestine in a mammal exposed to a trigger for LPS-induced bacterial translocation, the method comprising the step of administering an amount of TCF-II effective to reduce LPS-induced bacterial translocation, wherein the TCF-II is administered after exposure of the mammal to the trigger for LPS-induced bacterial translocation (claims 13-21) or administering an amount of TCF-II effective to reduce

Art Unit: 1645

LPS-induced bacterial translocation, wherein the TCF-II is administered after exposure of the mammal to the trigger for LPS-induced bacterial translocation (claims 28-30). The teachings of the specification are limited to Example 2, wherein two groups of mice were treated, the first group, the controls were intravenously administered vehicle and the rats in the second group were intravenously administered TCF-II at a time point 30 hours before LPS intravenous administration and were administered 3 times every 12 hours a dose of vehicle or 200ug/kg of TCF-II. 24 hours after the LPS administration the number of bacteria detected in the mesenteric lymph nodes were quantitated (see Figure 2) by means of colony formation. Figure 2, demonstrates that there was no significant difference in the number of bacteria between the treated and untreated groups of mice. In fact, the standard deviation bars as represented by the number of bacterial colonies on either Sheep serum medium or McConky medium overlap. This overlap is made clear by adding or subtracting the variation as represented by the number following  $\pm$  in Figure 2. Therefore, the specification does not teach that TCF-II was effective in reducing bacterial translocation as claimed, for TCF-II as administered 30 hours prior to LPS-administration and 3 times every 12 hours thereafter. Because the specification does not teach a statistically significant reduction with TCF-II as administered according to Example 2, then one skilled in the art would have reason to doubt, that pre-administration or post administration or concurrent administration with LPS-induction would provide for a reduction in LPS-induced bacterial translocation in any mammal. Additionally, the specification does not teach any "trigger" for LPS-induced bacterial translocation. Example 2, teaches that the administration of LPS itself allegedly induces bacterial translocation. As such, the skilled artisan would not know on what population to practice the claimed invention since, there are no triggers set forth in the specification that provide for LPS-induced bacterial translocation. In view of the lack of statistical relevance of the observations set forth for mice in Example 2 and Figure 2, one skilled in the art could not practice the invention as claimed.

Claims 2-5, 8-10, 13-16, 19-21, 24-27, 35 rejected under 35 U.S.C. 102(b) as being anticipated by Masunaga et al (U.S. Patent No. 5,714,461, issued Feb 3, 1998).

As to claims 2-5, 8-10, and 35, the claims are drawn to administering an amount of isolated or purified tissue cytotoxic factor-II (TCF-II) effective to reduce sepsis-associated lethality, wherein TCF-II is administered before the onset of sepsis in the mammal. Masuanga et al teach the administration of TCF-II by intravenous route in 10 mM phosphate buffer, pH 6.8-7.2, containing 0.01% Tween 80 and 0.25% human serum albumin and 0.15M sodium chloride (column 7, lines 1-5). Masunaga et al teach that the TCF-II may be administered by a variety of routes such as intravenous, intraarterial, intramuscular and subcutaneous (column 3, lines 35-41). Masunaga et al teach the administration of TCF-II prior to sepsis in normal rats. Because the development of sepsis can not be absolutely predicted, the administration of the TCF-II to the male rats is deemed to meet the limitation of administration prior to sepsis in mammal that develops sepsis and since the structurally identical compound is administered in the identical dosages and formulations (see Experiment 1, bridging columns 6-7) prior to sepsis. As such, the method as set forth in Masuanga et al would inherently provide for a reduction in sepsis-associated lethality.

As to claims 13-16 and 19-21, the claims are drawn to administering an amount of isolated or purified tissue cytotoxic factor II effective to reduce LPS-induced bacterial translocation, wherein TCF-II is administered to the mammal after the trigger for LPS-induced bacterial translocation. There are no triggers specifically taught by the specification. However, the specification teaches that bacterial translocation is observed wherein a patient is severely damaged by burn, major surgery etc. (specification page 2, third full paragraph). Masunaga et al teach the administration of TCF-II at and after rat surgery comprising 70% resection of the liver. The TCF-II is administered every 12 hours for 2 days (see Experiment 2, column 7). Because the structurally identical compound, using the same compositions and dosages of Masunaga is administered to a surgery patient

Art Unit: 1645

as is apparently contemplated by the specification and that the specification does not define a "trigger" as claimed herein, the method of Masunaga inherently provides for the claimed reducing LPS-induced bacterial translocation.

As to claims 24-27, the claim recites a method for reducing sepsis-associated lethality in a mammal exposed to a trigger for sepsis, the method comprising the step of administering an TCF-II prior to exposure of the mammal to the trigger for sepsis, within 48 hours of exposure of the mammal to the trigger, wherein the trigger is surgery, and at the time of exposure. The specification defines the transfer of bacteria or products secreted by them from the intestine is called bacterial translocation or endotoxin translocation and is noticed as a trigger of inducing sepsis (specification page 2, third full paragraph). As such, the administration of TCF-II to normal rats in Example 1, bridging column 6-7 anticipates this invention. Further, claim 24 is read to encompass administration after the trigger, because it does not recite within 48 hours "before" exposure to the trigger. As such, this claim stands anticipated Masuanga et al in view of the administration multiple times for 48 hours after surgery.

As to claims 31 and 33, the claims are drawn to methods for reducing sepsis-associated lethality in a surgery patient by administering to a surgery patient an amount of isolated or purified TCF-II effective to reduce sepsis-associated lethality. Masunaga et al teach the administration of TCF-II at and after rat surgery comprising 70% resection of the liver. The TCF-II is administered every 12 hours for 2 days (see Experiment 2, column 7). Because the art administers the structurally identical compound using the same composition and dosages and the TCF-II composition of Masunaga is administered to a surgery patient as claimed, the administration of the TCF-II in the method of Masunaga inherently provides for a reduction in sepsis-associated lethality as recited in the preamble of the claims.

Claims 13-16, 19-21, 31 and 33 are rejected under 35 U.S.C. 102(b) as being anticipated by Masunaga et al (Canadian Patent Application 2100720, Open to public inspection 01/17/94).

Masunaga et al is cited to teach subcutaneous and topical administration of TCF-II to surgical incisions at the time of surgery and thereafter. The Patent Application contemplates the same dosages, same composition and same routes of administration of the same patient population (i.e. surgery patients). Because the art administers the structurally identical compound using the same composition and dosages and the TCF-II composition of Masunaga is administered to the same patient population a surgery patient or trigger for LPS-induced bacterial translocation as claimed, the administration of the TCF-II in the method of Masunaga inherently provides for a reduction in sepsis-associated lethality or reduction of LPS-induced bacterial translocation as recited in the preamble of the claims.

#### *Status of Claims*

All claims stand rejected.


#### *Conclusion*

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Art Unit: 1645

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Patricia A. Duffy whose telephone number is 571-272-0855. The examiner can normally be reached on M-F 6:30 am - 3:00 pm. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Lynette Smith can be reached on 571-272-0864.

The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

  
Patricia A. Duffy

Primary Examiner

Art Unit 1645